Saudi Journal of Ophthalmology (2011) **25**, 97–98

King Saud University

Saudi Journal of Ophthalmology

www.saudiophthaljournal.com
www.ksu.edu.sa
www.sciencedirect.com

EDITORIAL

Changing paradigms in the treatment of diabetic retinopathy

The world is facing an epidemic of diabetes mellitus. Currently, more than 200 million people in the world have diabetes and it is predicted that this number will double in about 20 years. Diabetic retinopathy is the most common microvascular complication of diabetes and remains one of the leading causes of blindness worldwide among adults aged 20–74 years. The prevalence of diabetic retinopathy increases with the duration of diabetes, and nearly all persons with type 1 diabetes and more than 60% of those with type 2 have some retinopathy after 20 years. The two most important visual complications of diabetic retinopathy are diabetic macular edema and proliferative diabetic retinopathy.

Five large randomized controlled trials provided the scientific basis for care in the diabetic patient to preserve vision. The Diabetes Control and Complications Trial, the United Kingdom Prospective Diabetes Study, the Diabetic Retinopathy Study, the Early Treatment Diabetic Retinopathy Study and the Diabetic Retinopathy Vitrectomy Study demonstrated that strict metabolic control early in the course of diabetes, tight blood pressure control, panretinal photocoagulation, focal/grid laser photocoagulation, and early vitrectomy in patients with type 1 diabetes who had recent vitreous hemorrhage were effective at slowing the progression of retinopathy and reducing visual loss. However, due to the limitations of the current treatments, new therapeutic approaches are being developed. Intravitreal triamcinolone acetonide is reported to generate favorable results in the treatment of diffuse diabetic macular edema. However, the Diabetic Retinopathy Clinical Research Network demonstrated that focal/grid photocoagulation is a better treatment than intravitreal triamcinolone acetonide in eyes with diabetic macular edema. In addition, triamcinolone is associated with risks of elevated intraocular pressure and cataract. Anti-vascular endothelial growth factor agents are effective adjunctive treatment to laser photocoagulation or vitrectomy. Vitrectomy with the removal of the posterior hyaloid without internal limiting membrane peeling seems to be effective in eyes with persistent diabetic macular edema, particularly in those associated with vitreomacular traction (Abu El-Asrar and Al-Mezaine, 2010a). Our understanding of the role of the vitreous body in diabetic retinopathy has led investigators to use pharmacologic vitreolysis in the management of diabetic retinopathy. The agents used are purified ovine hyaluronidase (vitrase), autologous plasmin and plasmin. Pilot studies demonstrated that ovine hyaluronidase helps to clear vitreous hemorrhage. Autologous plasmin was a safe and effective adjunct to vitrectomy for diabetic macular edema and proliferative diabetic retinopathy. Intravitreal injection of autologous plasmin enzyme before surgery was useful in inducing pharmacologic posterior vitreous detachment that allowed a more complete and less traumatic posterior vitreous cortex removal with a smooth retinal surface. In addition, the proliferative membranes became softened and were easily peeled without retinal tears. Intravitreal injection of autologous plasmic enzyme without the performance of vitrectomy was found to induce posterior vitreous detachment and reduced macular thickening due to refractory diffuse diabetic macular edema (Abu El-Asrar and Al-Mezaine, 2010b). Other emerging therapies include long-term-lipid-lowering therapy with fenofibrate, renin–angiotensin system blockers, peroxisome proliferator-activated receptor gamma agonists, protein kinase C β inhibitors, islet cell transplantation, and micropulse subthreshold diode laser (Abu El-Asrar et al., 2009). A Better understanding of the underlying molecular and biochemical mechanisms involved in diabetic retinopathy will lead to the development of targeted therapeutic interventions. The microvasculature of the retina responds to hyperglycemia through a number of biochemical changes, including activation of protein kinase C, increased advanced glycation end products formation, polyol pathway and oxidative stress, and activation of the renin–angiotensin system. There is an accumulating body of evidence that inflammation and neurodegeneration play a prominent role in the pathogenesis of diabetic retinopathy (Abu El-Asrar et al., 2009). In this issue, the recent advances in the treatment of diabetic retinopathy are discussed.

Peer review under responsibility of King Saud University.
doi:10.1016/j.sjopt.2011.01.008

Production and hosting by Elsevier

References

Abu El-Asrar, A.M., Al-Mezaine, H.S., 2010a. Advances in the treatment of diabetic retinopathy. Discov. Med. 47, 363–373.

Abu El-Asrar, A.M., Al-Mezaine. H.S. 2010. Pharmacologic vitreolysis in diabetic retinopathy. Curr. Pharm. Biotechnol., Nov 12 (E-pub ahead of print).

Abu El-Asrar, A.M., Al-Mezaine, H.S., Ola, M.S., 2009. Pathophysiology and management of diabetic retinopathy. Expert Rev. Ophthalmol. 4, 627–647.

Ahmed M. Abu El-Asrar, MD, PhD
Associate Editor,
Saudi Journal of Ophthalmology,
Department of Ophthalmology, College of Medicine,
King Saud University, Old Airport Road, P.O. Box 245,
Riyadh 11411, Saudi Arabia
Tel.: +966 1 4775723; fax: +966 1 4775724

Available online 31 January 2011